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Mycobacterial Diseases

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Short Communication

An Overview of Immunological *Mycobacterium tuberculosis*

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DESCRIPTION

Mycobacterium tuberculosis immune responses are only partially effective; they drive the bacteria into a dormant state but rarely eliminate them. Unfortunately, *Mycobacterium tuberculosis* latent state is reversible, and reactivation tuberculosis is the source of the majority of transmission. Animal and human studies have yet to yield a complete picture of the mechanisms or correlates of immunity to *Mycobacterium tuberculosis* infection, or why immunity fails to eradicate the pathogen [1].

Innate immune cells

Mycobacterium tuberculosis is aerosol-transmitted and lives primarily, if not exclusively, in professional phagocytic cells in the lungs, such as macrophages, neutrophils, monocytes, and Dendritic Cells (DCs). The progressive accumulation of neutrophils, inflammatory monocytes, interstitial macrophages, and DCs in the lungs characterizes the early innate immune response to *Mycobacterium tuberculosis* in mice. As these cells are recruited, they become infected by the expanding mycobacterial population and form early granulomas [2]. In other infectious diseases, phagocytic cell recruitment restricts and even eliminates invading pathogens, whereas phagocyte recruitment to sites of mycobacterial infection benefits the pathogen during the early stages of infection by providing additional cellular niches for bacterial population expansion.

Mechanisms of innate immunity in TB

Multiple pattern-recognition receptors recognize *Mycobacterium tuberculosis* components during the innate immune stage. TLR2 has the most identified mycobacterial agonists among the Toll-like Receptors (TLRs), including lipoproteins (as many as 99), phosphatidylinositol mannans, and lipomannan. TLR9 also detects mycobacterial DNA and helps macrophages and DCs produce cytokines in *Mycobacterium tuberculosis*-infected mice. Although deletion of Tlr2 and Tlr9, alone or in combination, has no discernible effect on *Mycobacterium tuberculosis* control in mice, deletion of the gene encoding the shared TLR adaptor molecule MYD88 results in a lethal infection. This is most likely due to faulty signalling in response to IL-1 and IL-1, as such signalling is also dependent on MYD88. *Mycobacterium tuberculosis* is also recognized by members of the C-type Lectin Receptor (CLR) family, including DC-SIGN, dectin 1, the mannose receptor, and mincle [3]. Deletion of any of these CLR genes has little or no effect on infection progression, whereas deletion of the gene encoding the shared CLR adaptor molecule CARD9 is associated with accelerated mortality and excessive neutrophilic lung inflammation.

Reactivation TB

Reactivation of latent tuberculosis indicates progression to active, symptomatic disease, which is typically characterised by *Mycobacterium tuberculosis* shedding in respiratory secretions, particularly during coughing. Reactivation TB must be distinguished from re-infection with a different strain of bacteria, which can happen even in immunocompetent people. Except in geographical areas with extremely high TB prevalence, most cases of TB in adults are due to reactivation [4]. Through strain genotyping, one study clearly demonstrated that reactivation TB can occur decades after initial infection. Although only a minority of cases are attributable to well-characterized immune defects, reactivation TB is widely attributed to 'weakened' immunity.

CONCLUSION

Developing effective TB vaccines poses unique challenges that necessitate a better understanding of protective and pathological immune responses in TB. First, no clear correlates of protective immunity have been identified, particularly in humans, making surrogate end points insufficient for assessing TB vaccine efficacy. Second, while systematic research and selection of vaccine antigens has resulted in the development of promising candidate vaccines, the efficacy of these vaccines in preventing tuberculosis in a human population has yet to be determined. Third, tuberculosis may be unique in its use of immune responses to promote transmission.

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